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Axon Guidance Defects in NFATc2/3/4 Mutant Embryos

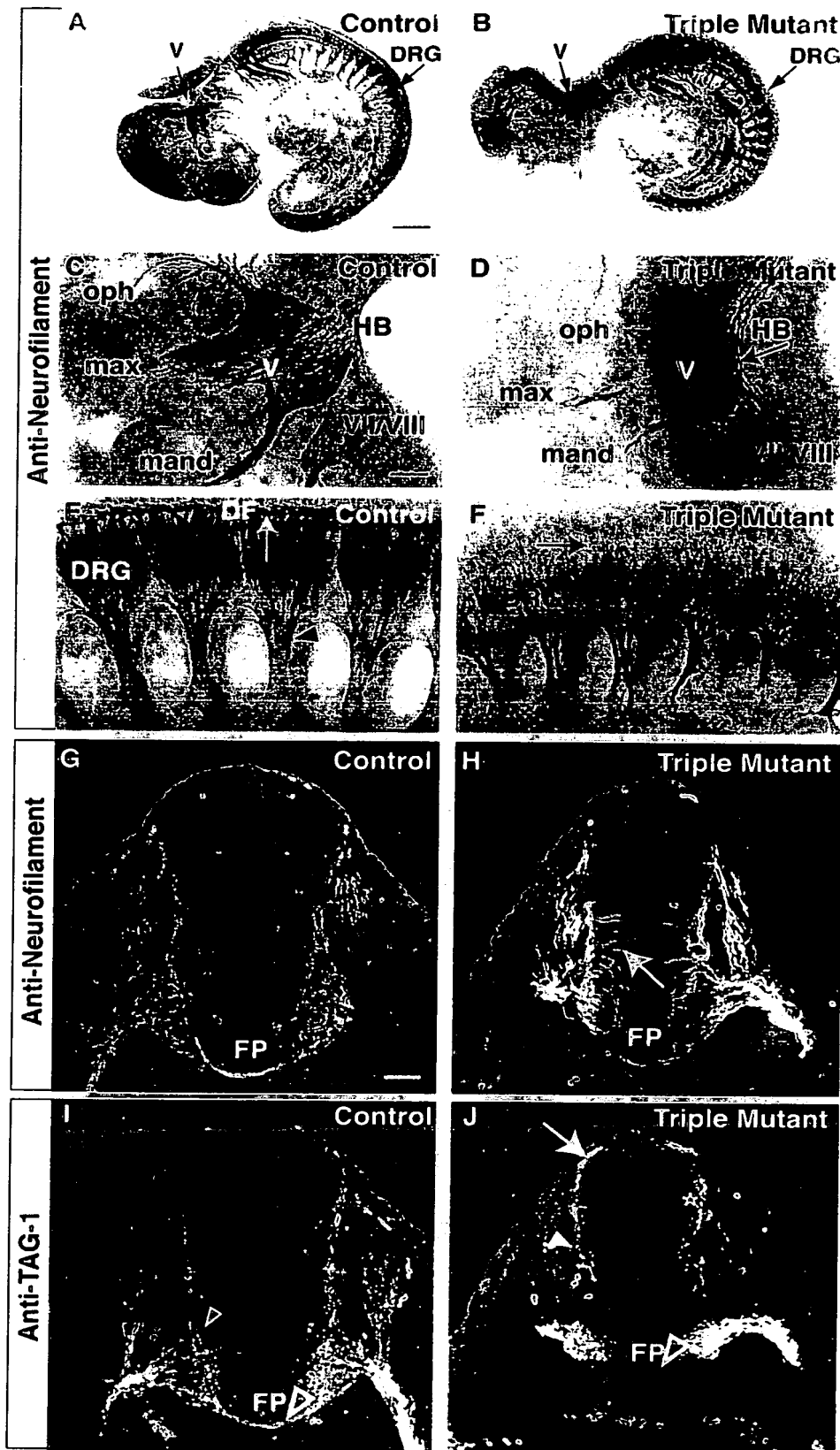


Figure 1 of 16

Pharmacological Calcineurin Inhibition during Embryonic Development Produces Defects Similar to those in NFATc2/c3/c4 Mutant Embryos.

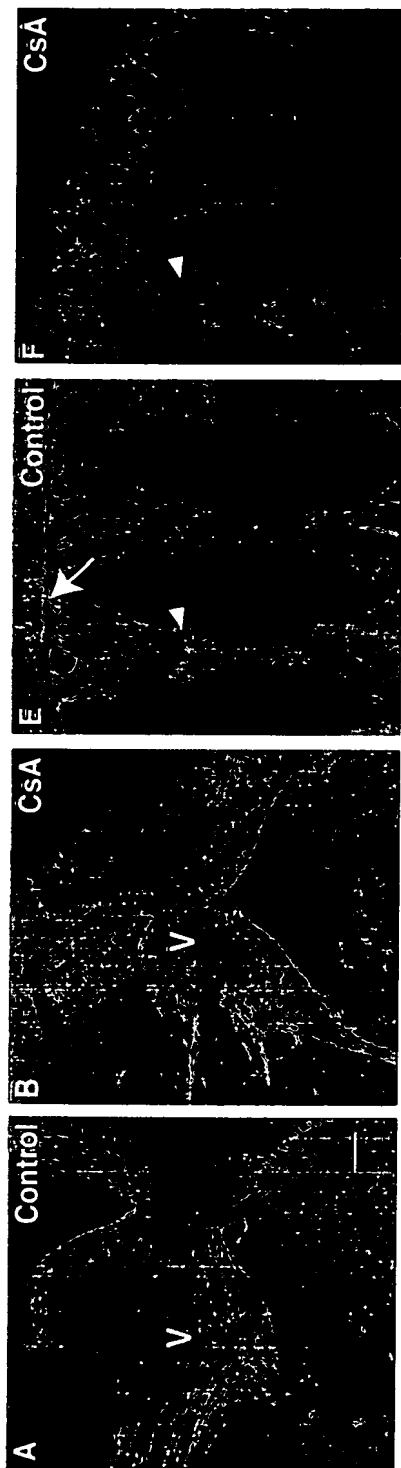
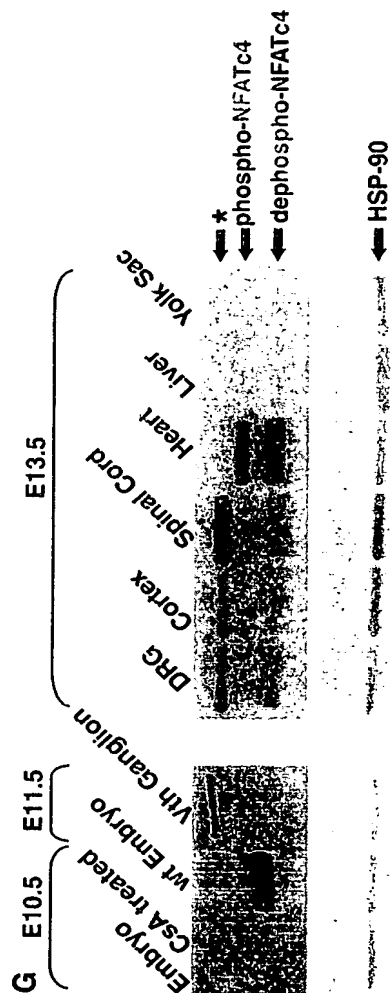


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Cell Autonomous Defect of Sensory Axon Growth

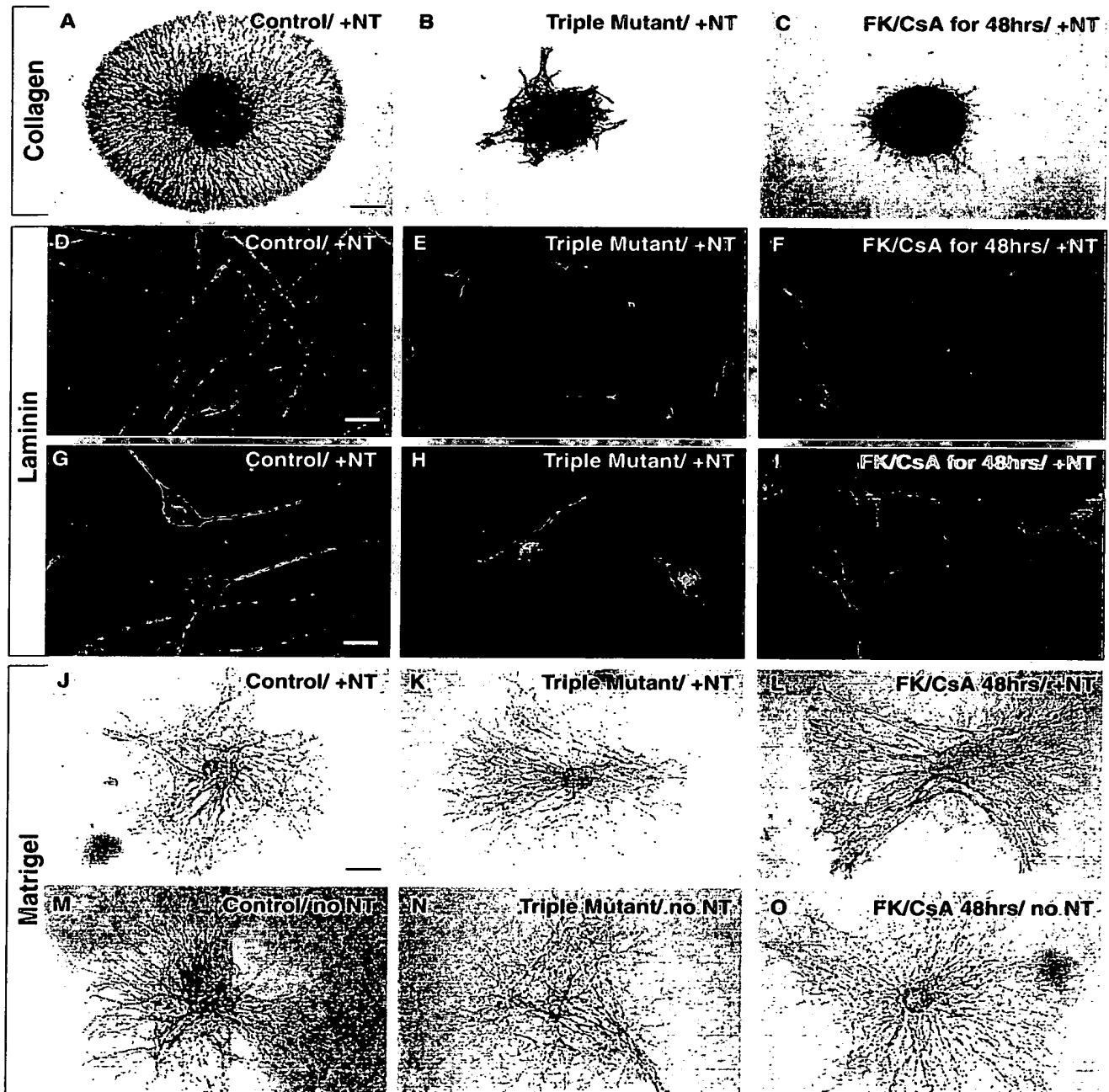


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Neither Calcineurin nor NFATc2/c3/c4 are Required for Sensory Neuron Survival *in vivo* or *in vitro*

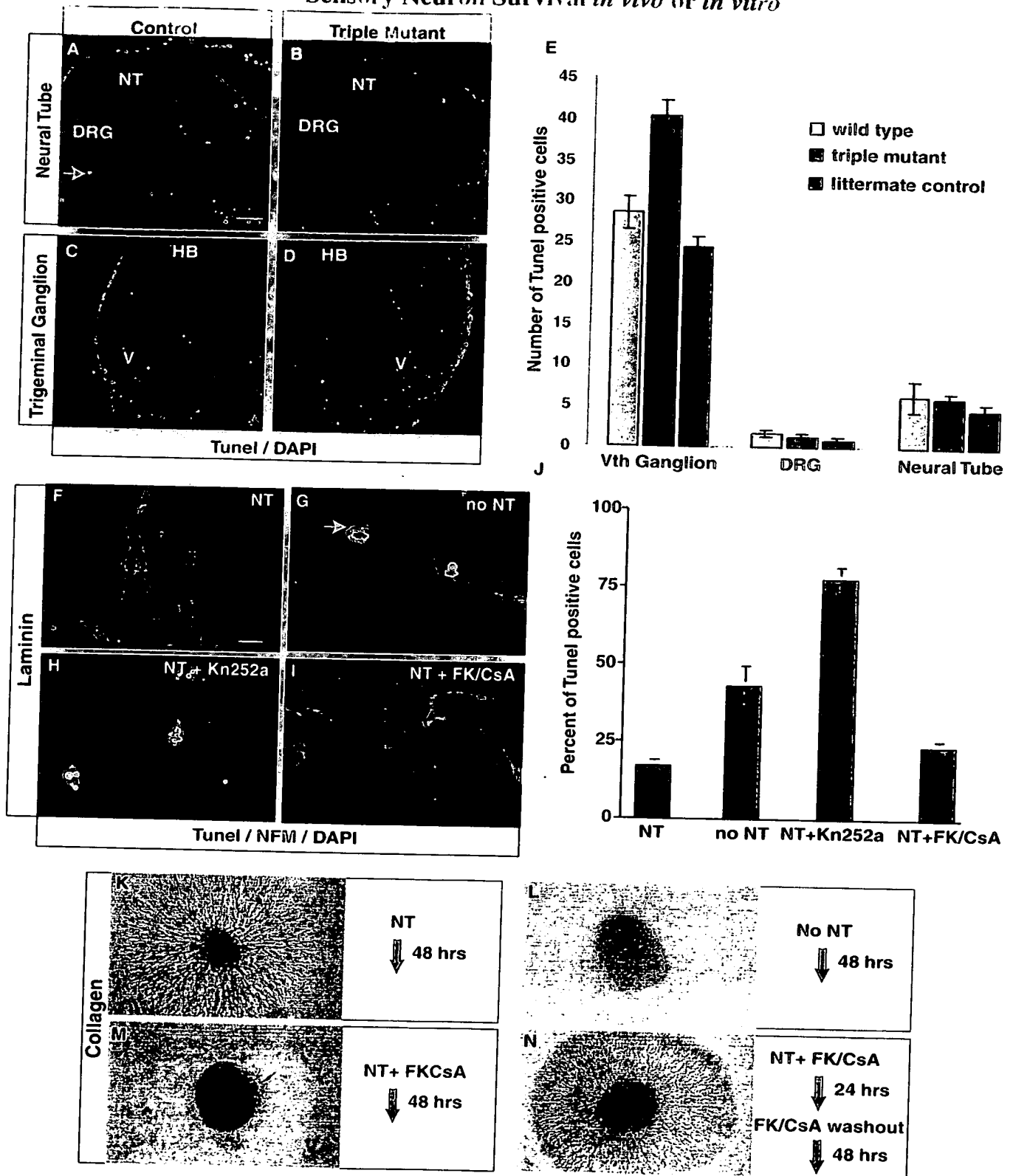
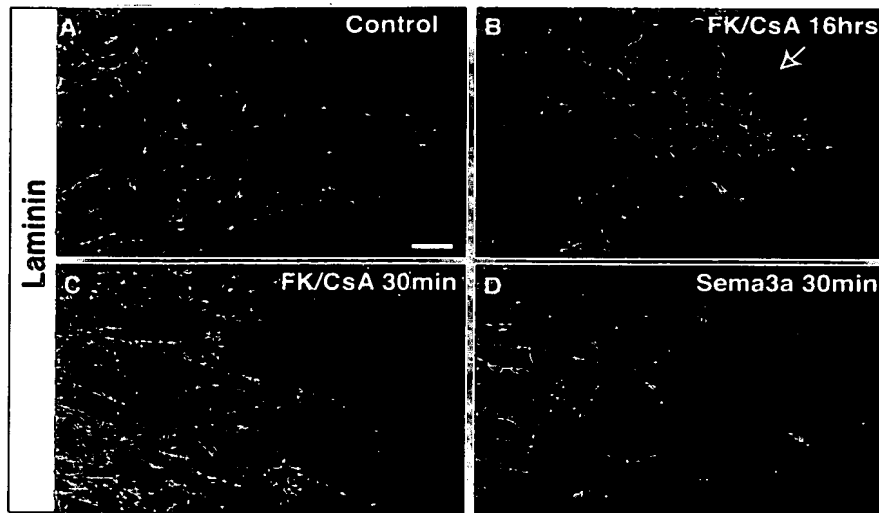


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**Calcineurin Inhibition does not Produce
Growth Cone Collapse**



**Neurite Outgrowth Arrest after Calcineurin Inhibition
Occurs with a Several Hour Delay**

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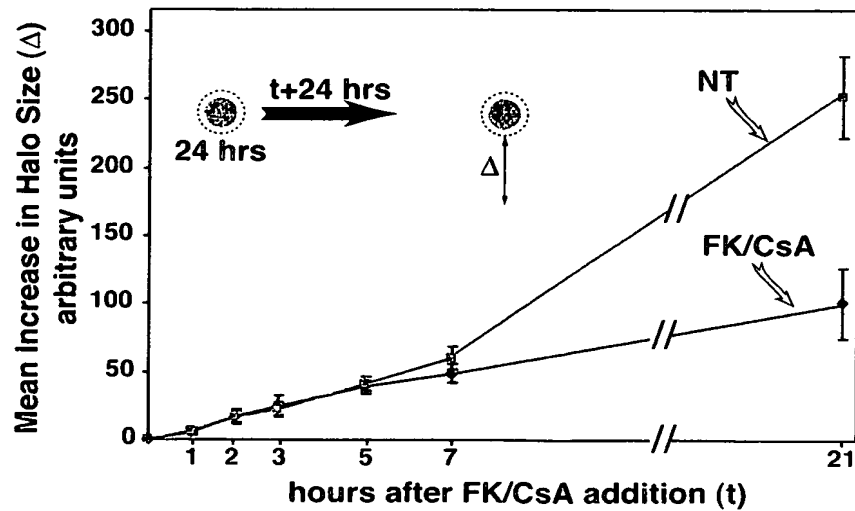


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Neurotrophins Regulate NFATc Translocation and Transcriptional Activation

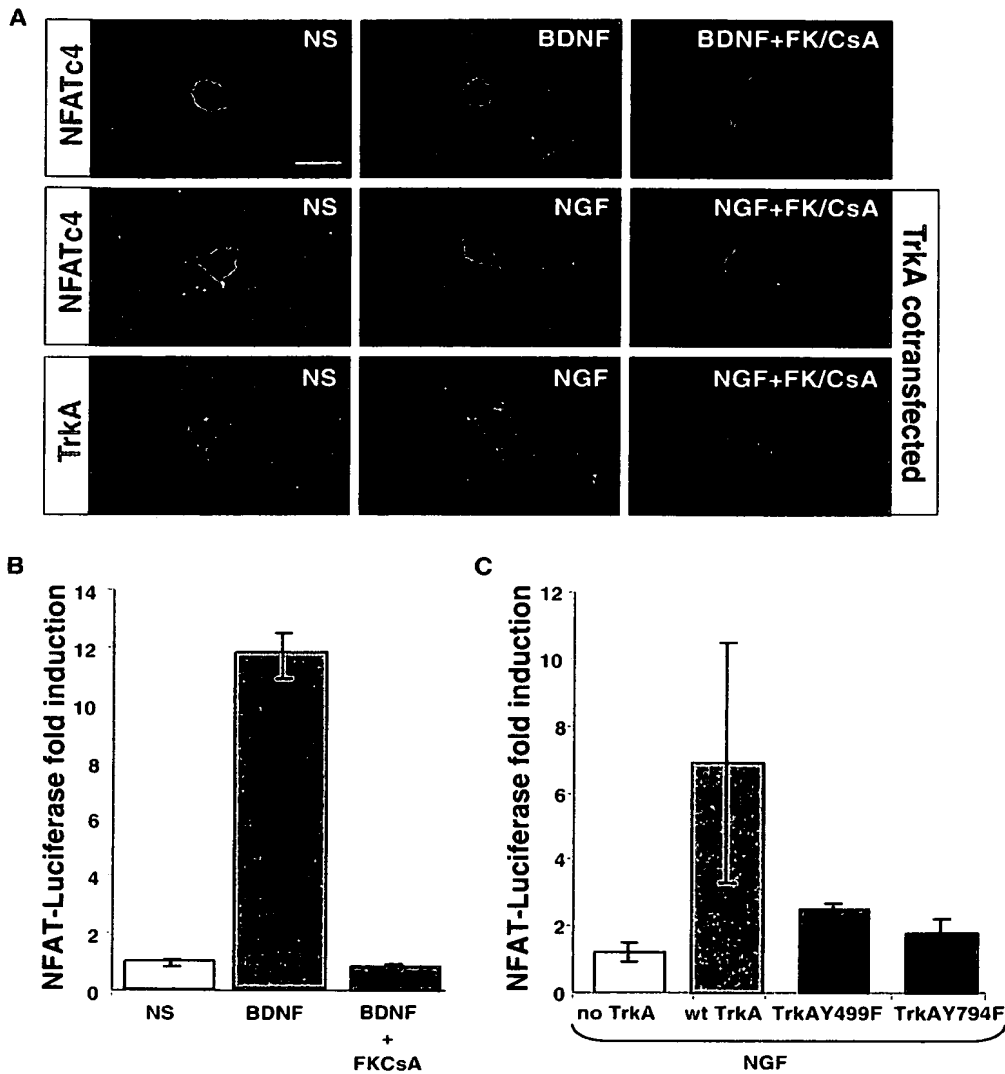


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**Inhibition of Calcineurin Blocks Netrin-dependent
Growth of Dorsal Spinal Cord Explants**

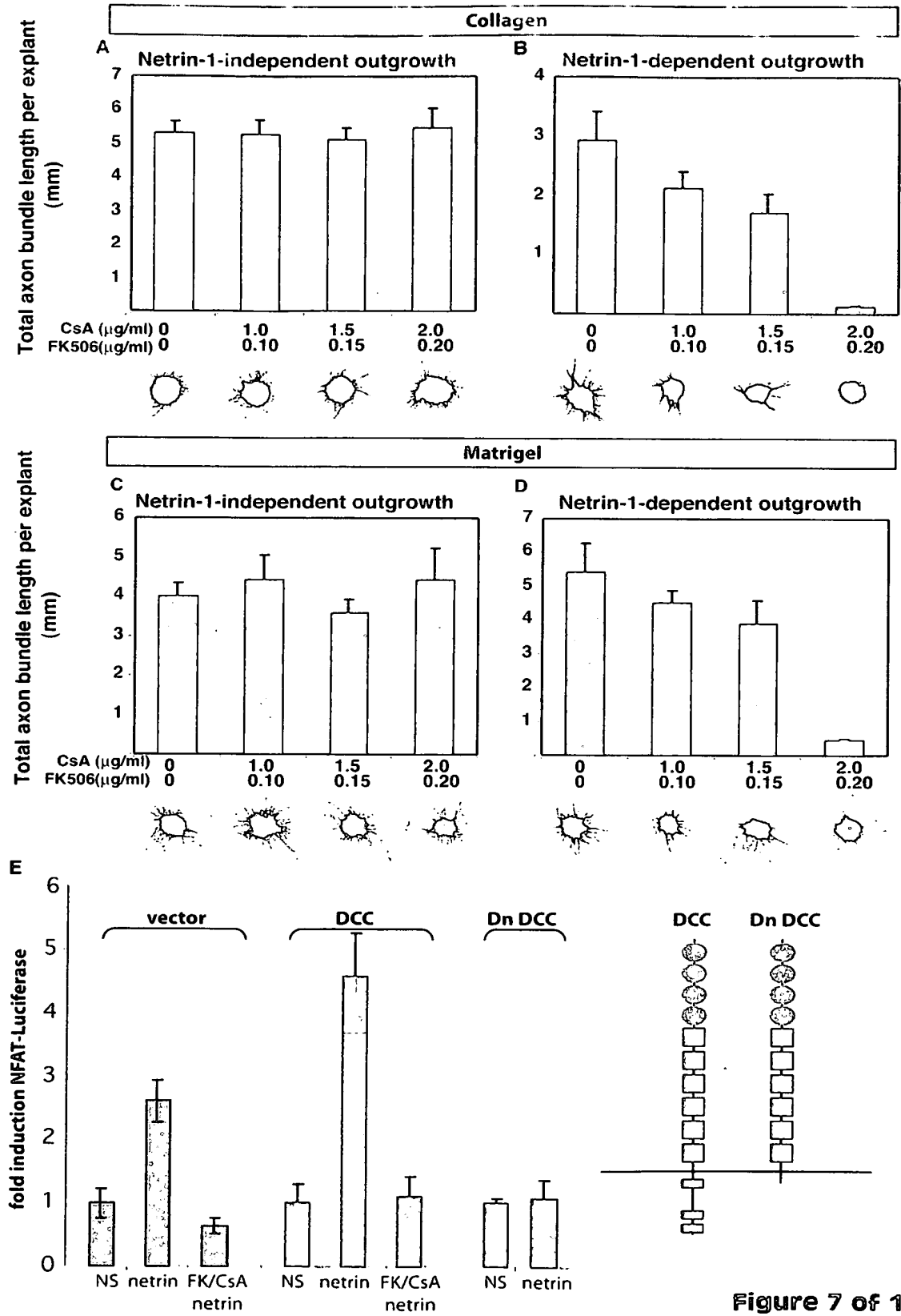


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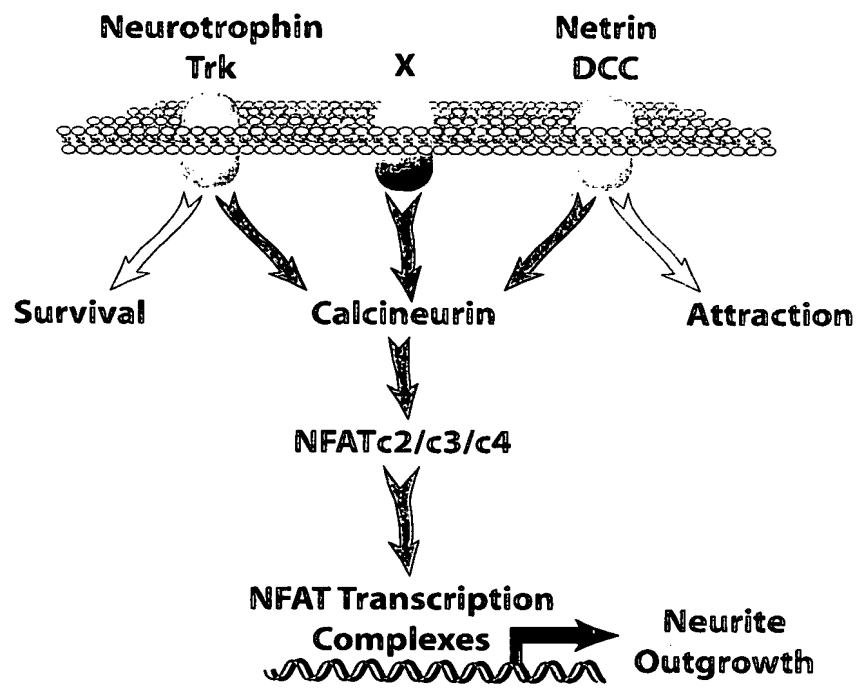


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Axon Guidance Defects in NFATc2/3/4 Mutant Embryos

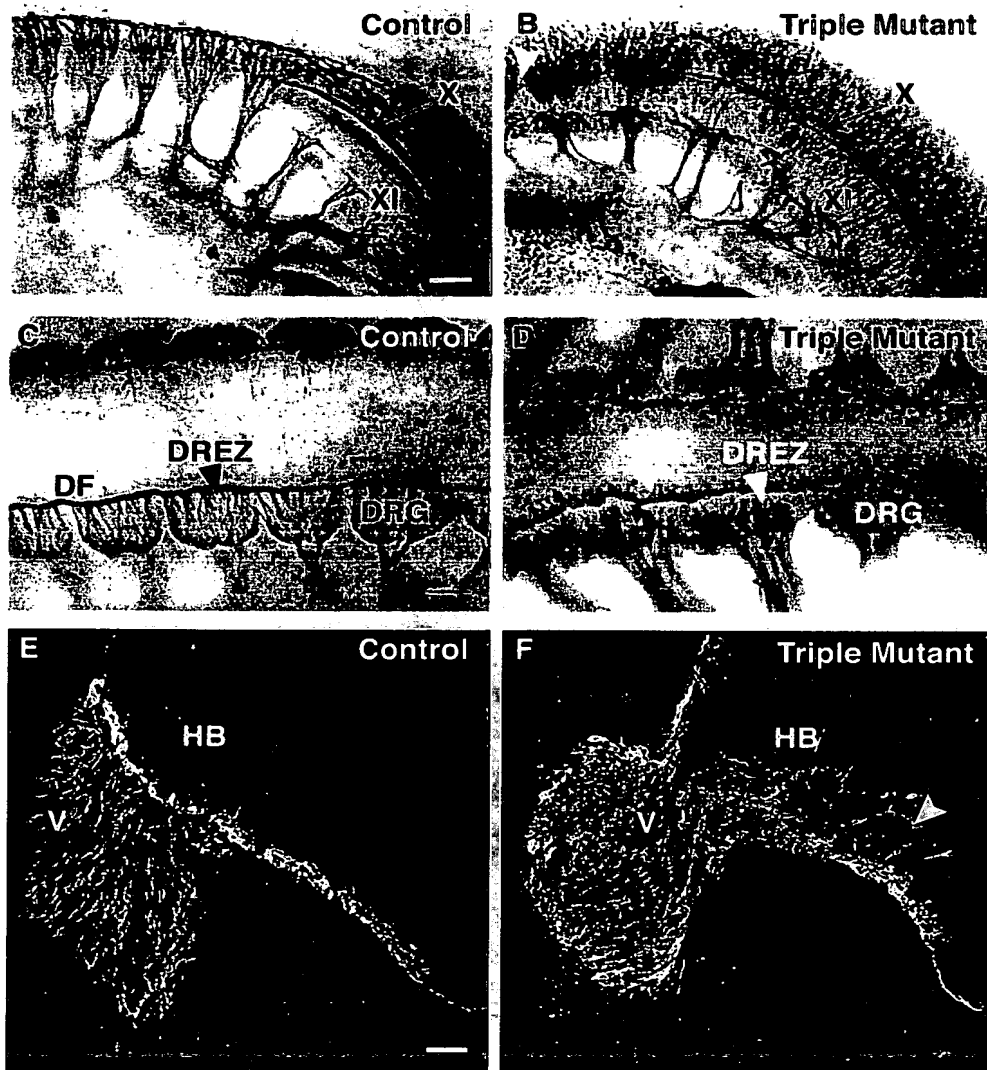


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Normal Cell Type Differentiation of NFATc2/c3/c4 Mutant Neurons

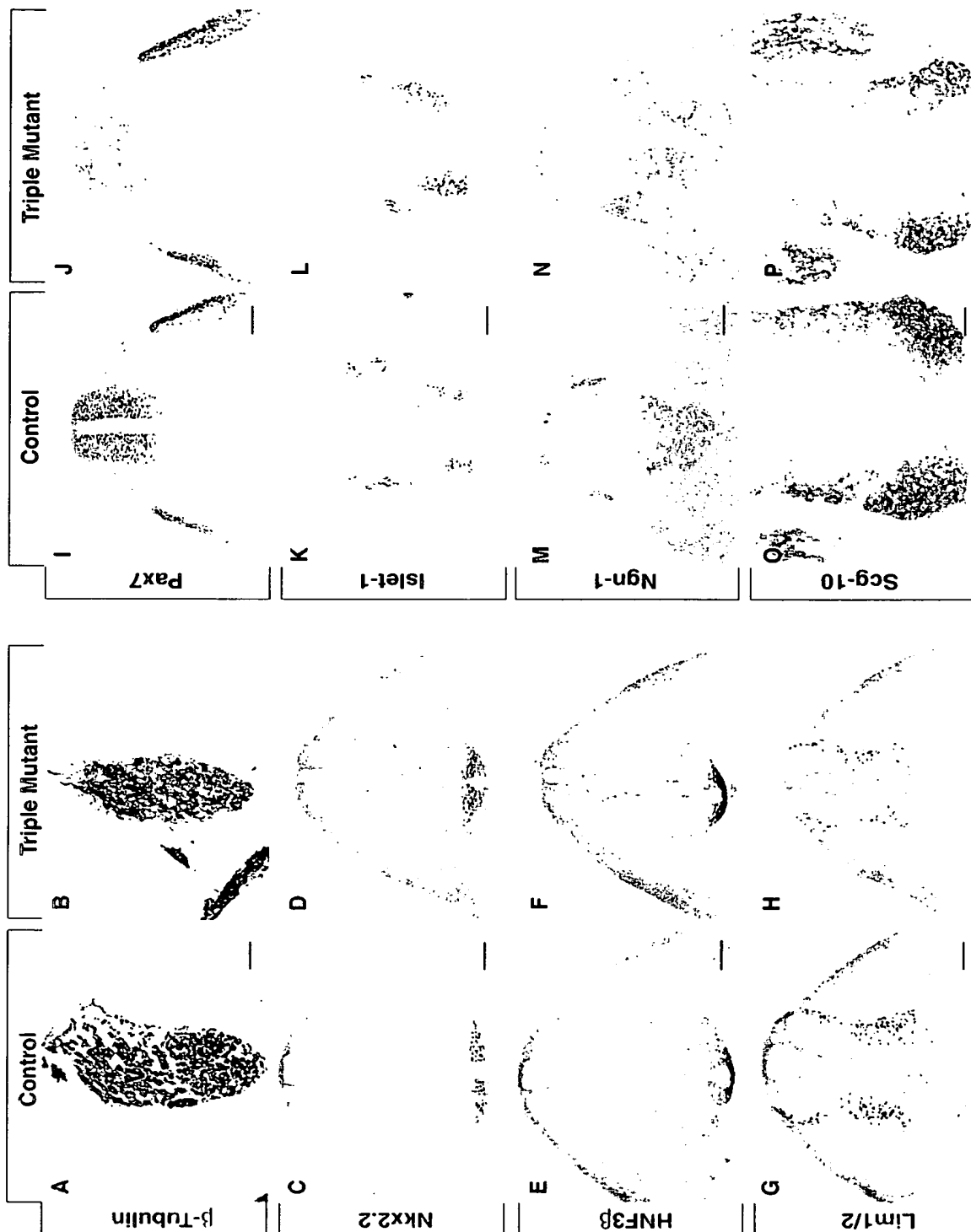


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Inhibition of the NFATc/Calcineurin Pathway Does Not Affect
Semaphorin-Induced Growth Cone Collapse

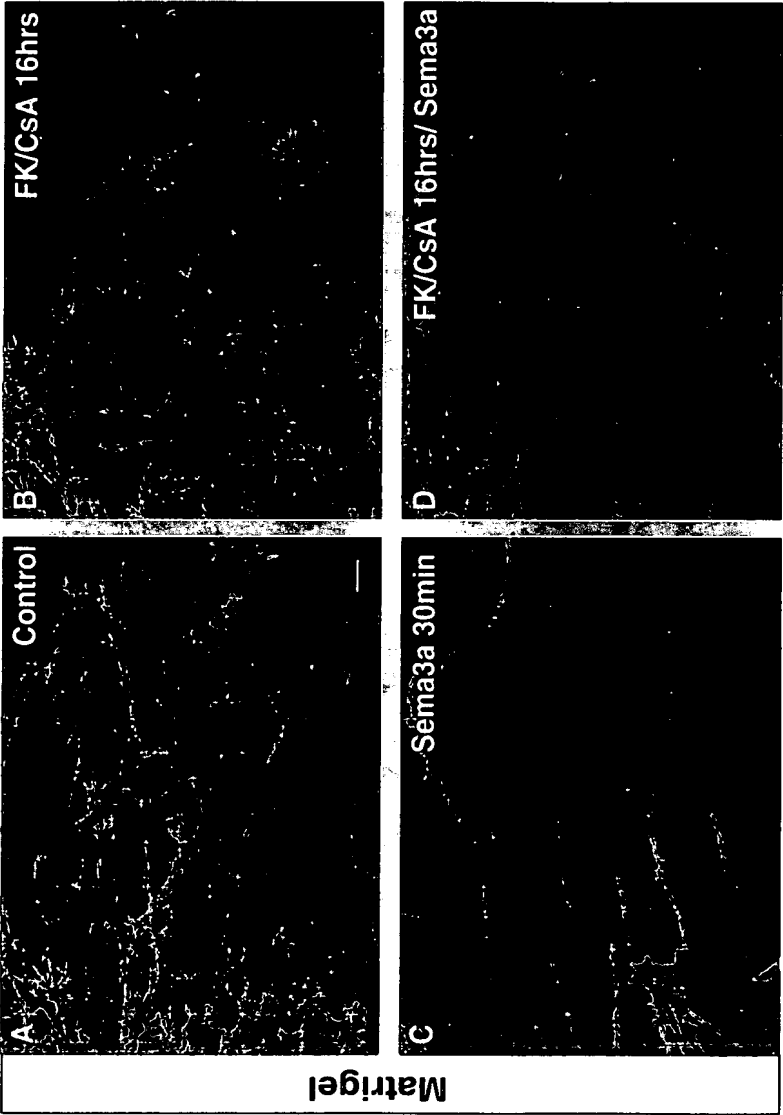


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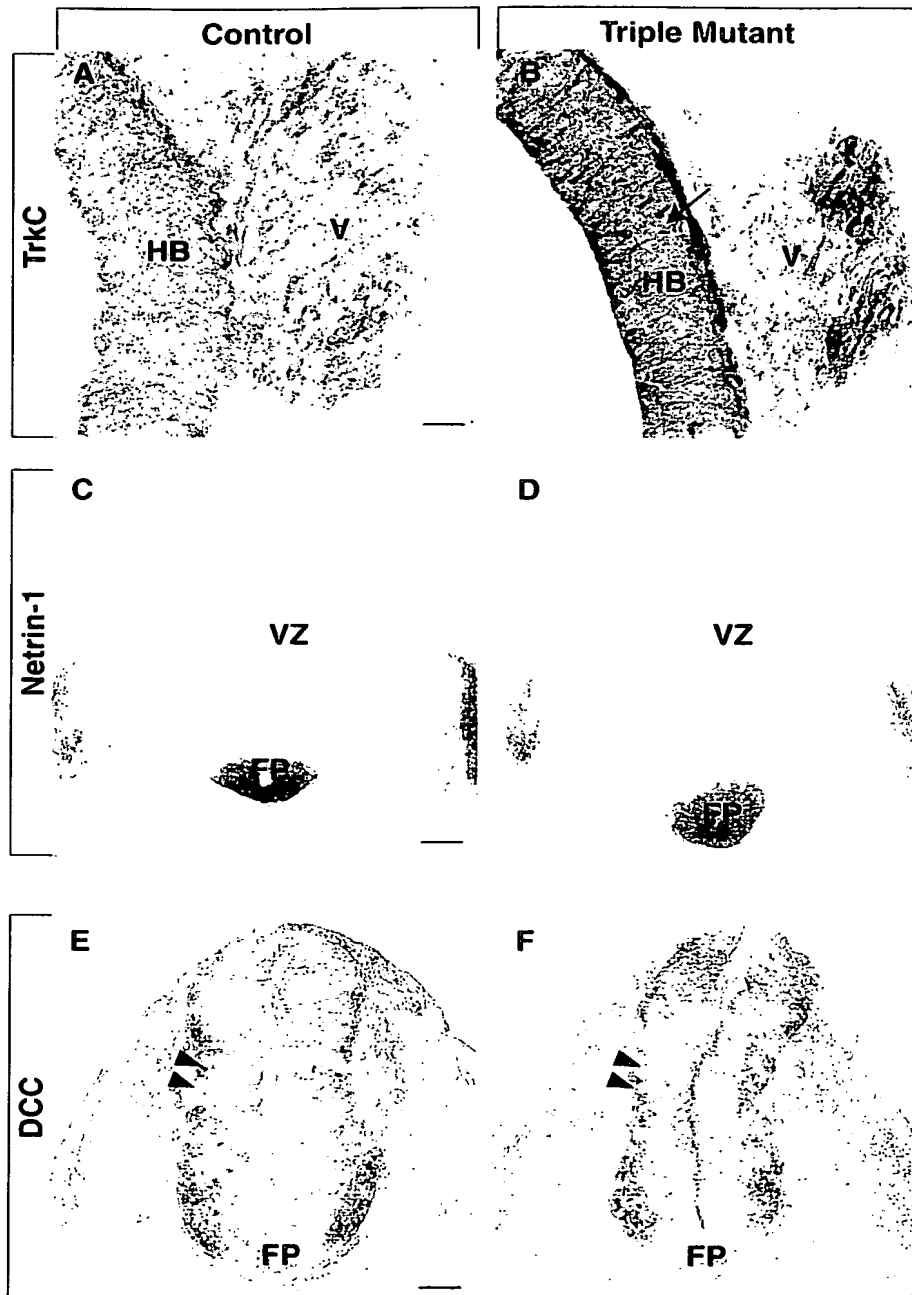


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